RESIDUAL ISCHEMIA AT PRE-DISCHARGE STRESS TEST FOLLOWING SYSTEMIC THROMBOLYSIS: THE GISSI-2 IRES RESULTS.

The GISSI-2 IRES (Ischemia Residua) Study Group.

The IRES study prospectively enrolled 454 unselected GISSI-2 pts aged ≤70 yrs 24 hrs following their first MI; 386 of them underwent a predischarge,off drug symptom-limited exercise test (ET) and in 360/386 (93%) pts a pre-discharge coronary angiography was also performed. A positive ET was found in 104 (27%) pts (angina in 31 and/or >1mm ST depression in 87), unrelated to sex,age, Q/non Q MI, thrombolytic agent (52% SK, 48% tPA) and time (1-6 hrs) of administration. A greater incidence of positive ET was found following inferior (32%) vs anterior MI (18%,p<0.05) and in pts with multiple (47%) vs single (27%,p<0.01) >70% coronary stenosis. The 6-month follow-up was completed in 311/320 (97%) pts with diagnostic (positive/negative) ET:CABG or PTCA were carried out in 36% of positive and 3.7% negative ET(p<0.01).Out of 278 medically treated pts, cardiac events (CE:4 deaths,13 non-fatal re-MI and 30 angina) occurred in 32% positive vs 12% (p<0.001) negative ET (angina: p<0.001; death and re-MI:NS).CE occurred in 35% of pts with positive and in 15% with negative ET following anterior MI (NS) and in 31% positive vs 9.6% negative ET following inferior MI (p<0.01). In 201 pts with single CAD,CE occurred in 17% positive vs 8% negative ET (NS); in 92 pts with multivessel CAD,CE were found in 50% positive vs 9% negative ET (p<0.001). Preliminary GISSI2-IRES results confirm,even following thrombolysis, the prognostic value of a positive ET in pts with inferior MI and/or multiple CAD.

THE HEMODYNAMIC EFFECTS OF CORONARY REPERFUSION IN RIGHT VENTRICULAR INFARCTION

Steven C. Ajluni,M.D., Joseph G. Samyn,M.D., Craig Neuner,M.D., David G. Rizik,M.D., William W. O'Neill,M.D., FACC, William Beaumont Hospital, Royal Oak, Michigan

Myocardial infarction (MI) accompanied by acute RV dysfunction produces unique hemodynamic sequelae and may portend a worse prognosis. The hemodynamic effects of reperfusion in this condition are not well understood. We studied 27 cases of RV MI over a 16 month period. Inclusion criteria required 1) an acute inferior wall MI by ECG, 2) an elevated RA pressure ≥ 10 mmHg, 3) noninvasive studies suggesting RV dysfunction. All pts underwent right and left heart catheterization. Group A consisted of 13 pts who underwent successful mechanical or thrombolytic reperfusion, and Group B, of 14 pts who did not demonstrate clinical reperfusion. Hemodynamics (RA and pulmonary capillary wedge pressure (PCWP)) were measured at baseline, and again at 8 hours.

RESULTS	Group A (n=13)	Group B (n=14)
Mean age (years)	62.8	70.8*
MAP ≤ 80 mmHg***	5/13	8/14
Multivessel disease	6/13	8/14
Mean CK peak (I.U.)	2094	1786
Initial RA (mmHg) (mean)	15.6	13.8
RA (8 hrs) (mmHg) (mean)	8.0	13.3**
Change in RA (mmHg) (mean)	-7.4	-0.6**
Initial PCWP (mmHg) (mean)	15.8	12.6
PCWP (8 hrs) (mmHg) (mean)	10.1	13.0*
Change in PCWP (mean)	-6.0	+0.3*
Survival until discharge	12/13 (92%)	9/14 (64%)

* p ≤ 0.05 ** p ≤ 0.01
*** mean arterial pressure at presentation
Variables such as measurement of fluid balance, use of intravenous nitroglycerin or inotropic agents, and use of an IABP did not differ significantly between the groups.
Conclusion: Successful reperfusion in the setting of RV MI correlates with rapid hemodynamic improvement and a trend toward improved survival.

EFFECTS OF SARUPLASE AND OF ALTEPLASE ON HEMOSTASIS IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION. A SINGLE-BLIND, RANDOMIZED TRIAL

Philippe Vanhove, and the Belgian Saruplase Alteplase Trial Group, Center for Thrombosis and Vascular Research, University of Leuven, Leuven, Belgium

The effects of saruplase (S) and alteplase (A) on hemostasis were compared in patients with acute myocardial infarction treated randomly, either with intravenous (i.v.) S (bolus of 20 mg, then 60 mg in 1 h; n=24) or with i.v. A (bolus of 10 mg, then 50 mg in 1 h and 40 mg in 2 h; n=28). I.v. heparin was given in all patients.

		Before treatment	End of treatment	End +1 h	End +8 to 24 h
Fibrinogen* (g/l) (Fg)	S	2.35	0.90	0.50	1.08
	A	2.50	1.80	1.90	1.95
Plasminogen* (%) (Plg)	S	88	28	21	34
	A	88	47	50	59
α_2-Antiplasmin*(%) (α_2-AP)	S	100	28	50	68
	A	102	52	83	87
FDPs*+ (µg/ml)	S	1.5	114	368	123
	A	1.9	27	55	29
D-dimer* (µg/ml)	S	0.30	2.76	4.21	1.65
	A	0.14	3.49	4.56	0.95

*Values represent medians. +Fibrin(ogen) degradation products.
Bleeding was observed in 5 and 8 patients of the S and A groups, respectively (not significant).
Conclusion: Posttreatment Fg, Plg and α_2-AP levels were significantly higher and FDPs significantly lower in the A group than in the S group. Posttreatment D-dimer levels were not significantly different between treatment groups. S induced more extensive hemostatic breakdown than A. In contrast, bleeding complications tended to be more frequent in the A group.

TEMPORAL DISTRIBUTION OF TREATMENT TO REPERFUSION TIMES IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION RECEIVING STREPTOKINASE OR TISSUE PLASMINOGEN ACTIVATOR.

Prediman K. Shah, M.D., Allan S. Lew, M.D., Bojan Cercek, M.D., William Ganz, M.D. Cedars-Sinai Medical Center, and University of California, Los Angeles, CA.

The 24 hr reperfusion (REP) rates in pts with acute myocardial infarction (AMI) are similar with tissue plasminogen activator (TPA) and Streptokinase (SK) despite a significally higher REP with TPA at 90 mins. This indicates that REP occurs frequently >90 mins after start of SK. To examine this possibility we reviewed 66 pts treated with SK and 44 pts treated with TPA who manifested signs of successful REP and were carefully observed for upto 3 hours after treatment. Creatine kinase (CK) levels and ECG's were taken at 15-30 min intervals. REP was diagnosed when ST segment elevation resolved rapidly (by ≥ 50% within 15±10 mins) and CK increased abruptly (by ≥13% of peak in the first hour). The diagnostic value of rapid CK rise for REP was previously angiographically validated during an intracoronary SK study. Time of REP was defined as the time of onset of the resolution of ST segment elevation. The time to REP averaged 55 ± 33 mins in the SK and 48 ± 22 mins in the TPA group (p=NS). The time to REP was >90 mins (Range:95-140 mins) in 13/66 (20%) SK patients and in only 1/44 (2%) TPA pts (p=0.01). Conclusion: Our data show that in a significant proportion (20%) of pts with AMI receiving SK, reperfusion occurs > 90 mins but < 150 mins after start of treatment, probably still early enough to affect myocardial salvage. This observation may, at least in part, explain the similarity of early mortality rates despite a significant difference in REP rates at 90 mins after start of treatment.